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Prevalence of *Toxoplasma gondii* Antibodies in Domestic Cats From Rural Ohio

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ABSTRACT: Antibodies to *Toxoplasma gondii* were determined in serum samples from 275 domestic cats from a mobile spay and neuter clinic from 8 counties in Ohio. The modified agglutination test incorporating whole formalinized tachyzoites and mercaptoethanol was used to determine antibodies. Antibodies to *T. gondii* were found in 133 (48%) out of 275 cats: in titers of 1:25 in 24, 1:50 in 37, and 1:500 or more in 72. The highest prevalence (62% of 78) was in outdoor cats. The prevalence of *T. gondii* antibodies in 48% of cats suggests widespread contamination of the rural environment with oocysts.

Cats are essential for the maintenance of *Toxoplasma gondii* infections in animals and humans because they are the only hosts that can excrete the environmentally resistant oocysts (Dubey and Beattie, 1988). Cats may become infected with *T. gondii* by ingesting infected tissues or by ingesting food and water contaminated with oocysts or transplacentally. Epidemiologic data and results of feeding on infected tissues or oocysts indicate that cats probably acquire infection in nature by ingesting infected tissues. The prevalence of *T. gondii* infection in indoor cats is lower than in feral outdoor cats (Dubey, 1973; Dubey and Beattie, 1988). It has been more than 20 yr since a prevalence of *T. gondii* was determined in Ohio cats (Claus et al., 1977). In the present paper, serologic prevalence of *T. gondii* in 275 domestic cats from 8 counties in Ohio is reported.

Serum samples were obtained from cats in a mobile spay and neuter clinic while the cats were under general anesthesia for surgery. The cats were presented to the mobile spay and neuter clinic in different areas of 8 Ohio counties, sometimes rural and sometimes urban. The rural counties included Delaware, Medina, Miami, Muskingum, Licking, and Preble. Urban counties included the city of Columbus in Franklin county and the city of Dayton in Montgomery county. The cat populations were divided into barn cats, feral cats, and outside or in–out cats. The barn cats were those that lived in barns and were handled by the owners of the farms but were not necessarily vaccinated. The feral cats were those that had to be caught with nets, were not vaccinated, and not spayed or neutered. The outside or in–out cats were those that were handled, lived inside the house or outside, and were up-to-date in their vaccinations. Cats were anesthetized using isoflurane (Vedco, St. Joseph, Missouri). Once anesthetized, 3 ml of blood was collected through a jugular venipuncture, and samples were centrifuged and stored at −80 C.

Sera were transferred from Columbus, Ohio, to Beltsville, Maryland, where serologic examination was performed. Sera were diluted 1:25, 1:50, and 1:500 with phosphate buffer saline and tested by the modified agglutination test (MAT), as described by Dubey and Desmonts (1987). Whole formalin-fixed tachyzoites and mercaptoethanol were used in the antigen for MAT. On the basis of extensive evaluation in cats fed tissue cysts, a titer of 1:25 was considered indicative of *T. gondii* infection in cats (Dubey and Thulliez, 1989; Dubey et al., 1995; Dubey et al., 1995a, 1995b).

Antibodies to *T. gondii* were found in 133 out of 275 (48%) cats: in titers of 1:25 in 24, 1:50 in 37, and 1:500 or more in 72. Thus, 54 % of seropositive cats had high (≥1:500) antibody titers (Table I). Seropositive cats were found in all 8 counties examined (Table I).

Antibodies to *T. gondii* were found in 53 out of 115 (46%) males and 75 out of 150 (50%) females; sex was not recorded for 5 out of 10 seropositive cats. The owners were questioned whether the cats were totally indoors, were totally outdoors, spent most of the time in the barn, or were caught wild by the owner; the information provided is summarized in Table II. Whereas the reliability of the data depends on what the owner provided, feral cats had the highest prevalence (62%).

Seroprevalence of *T. gondii* in cats varies depending on the type of cats (feral vs. domestic), age of cats, method of serologic testing, and geographic location; worldwide serologic findings up to 1987 were summarized earlier (Dubey and Beattie, 1988). In the previous study, of 1,000 cats surveyed from a humane shelter in Columbus, Ohio, in 1975, antibodies to *T. gondii* were found in 39%, and antibodies were measured by the indirect fluorescent antibody test (IFAT) (Claus et al., 1977). Of these 1,000 cats, 7 were found to have *T. gondii* oocysts in their feces (Dubey et al., 1977). Because the period of time these cats were in the humane shelter was unknown, it is not certain whether the cats became infected in the shelter or in the homes from which they came.

In another study on *T. gondii* infection in animals on swine farms in Illinois, 267 out of 391 (68%) cats trapped had antibodies to *T. gondii* (Dubey et al., 1995). Another study reported prevalence in 41.9% of 74 cats from swine farms in Iowa (Smith et al., 1992). The same serologic test (MAT) was used in the present study and in studies on Illinois and Iowa cats, whereas a different serological test was used in the Ohio study (IFAT). These data indicate a high endemicity of *T. gondii* in the rural Midwest because all seropositive cats had probably already shed oocysts and contaminated the environment; cats can shed millions of oocysts after ingesting few bradyzoites (Dubey, 2001). Because oocysts

TABLE I. Prevalence of antibodies to *Toxoplasma gondii* from cats from 8 counties in Ohio.

County	Total no. of cats	Seropositive cats (≥1:25) Total	Percent	No. of cats with antibody in titers 1:25*	1:50*	≥1:500*
Delaware	21	15	71	1	5	9
Franklin	86	27	31	6	7	14
Medina	28	22	79	2	1	19
Miami	25	15	60	0	6	9
Montgomery	65	32	49	12	8	12
Muskingum	7	1	14	0	1	0
Licking	25	14	56	1	5	8
Preble	18	7	39	2	4	1
Total	275	133	48	24	37	72

* Serum dilution.

TABLE II. Seroprevalence of *Toxoplasma gondii* antibodies in cats according to habitat.

Habitat	Total	% Seropositive (≥1:25)	No. of cats with antibody in titers 1:25*	1:50*	≥1:500*
Barn	94	50	5	15	27
Outside	80	38	9	9	13
Feral	78	62	7	11	30
Not recorded	23	44	3	2	2

* Serum dilution.

are rarely found in feces of cats, serologic prevalence data in cats are important for the determination of epidemiologic significance of *T. gondii* infection (Dubey et al., 1995). At any given time only about 1% of cats were found shedding *T. gondii* oocysts (Dubey and Beattie, 1988).

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LITERATURE CITED

CLAUS, G. E., E. CHRISTIE, AND J. P. DUBEY. 1977. Prevalence of *Toxoplasma* antibody in feline sera. Journal of Parasitology **63:** 266.

DUBEY, J. P. 1973. Feline toxoplasmosis and coccidiosis: A survey of domiciled and stray cats. Journal of the American Veterinary Medical Association **162:** 873–877.

———. 2001. Oocyst shedding by cats fed isolated bradyzoites and comparison of infectivity of bradyzoites of the VEG strain *Toxoplasma gondii* to cats and mice. Journal of Parasitology **87:** 215–219.

———, AND C. P. BEATTIE. 1988. Toxoplasmosis of animals and man. CRC Press, Boca Raton, Florida, 220 p.

———, E. CHRISTIE, AND P. W. PAPPAS. 1977. Characterization of *Toxoplasma gondii* from the feces of naturally infected cats. Journal of Infectious Diseases **136:** 432–435.

———, AND G. DESMONTS. 1987. Serological responses of equids fed *Toxoplasma gondii* oocysts. Equine Veterinary Journal **19:** 337–339.

———, M. R. LAPPIN, AND P. THULLIEZ. 1995a. Diagnosis of induced toxoplasmosis in neonatal cats. Journal of the American Veterinary Medical Association **207:** 179–185.

———, ———, AND ———. 1995b. Long-term antibody responses of cats fed *Toxoplasma gondii* tissue cysts. Journal of Parasitology **81:** 887–893.

———, AND P. THULLIEZ. 1989. Serologic diagnosis of toxoplasmosis in cats fed *Toxoplasma gondii* tissue cysts. Journal of the American Veterinary Medical Association **194:** 1297–1299.

———, R. M. WEIGEL, A. M. SIEGEL, P. THULLIEZ, U. D. KITRON, M. A. MITCHELL, A. MANNELLI, N. E. MATEUS-PINILLA, S. K. SHEN, O. C. H. KWOK, AND K. S. TODD. 1995. Sources and reservoirs of *Toxoplasma gondii* infection on 47 swine farms in Illinois. Journal of Parasitology **81:** 723–729.

SMITH, K. E., J. J. ZIMMERMAN, S. PATTON, G. W. BERAN, AND J. T. HILL. 1992. The epidemiology of toxoplasmosis on Iowa swine farms with an emphasis on the roles of free-living mammals. Veterinary Parasitology **42:** 199–211.